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Case Report

The Psychiatric Masquerade of a Frontal Lobe Lesion: Lessons from a Late-Onset Case

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Abstract

Background: Neuropsychiatric symptoms are frequently encountered in dementia and may include delusions, hallucinations, and behavioral disturbances. However, the coexistence of factitious disorder (previously termed Munchausen syndrome) in elderly patients with dementia is exceedingly rare. The presence of uncontrolled diabetes mellitus further complicates the clinical course, influencing both cognitive decline and therapeutic decisions.
Case Presentation: We report the case of a 68-year-old man with uncontrolled type 2 diabetes mellitus who presented with persecutory delusions, fluctuating cognitive impairment, and repeated fabrication of seizure-like episodes. Clinical findings were incongruent with reported symptoms. Laboratory investigations revealed poor glycemic control, while neuroimaging demonstrated only mild cerebral atrophy. The inconsistency between subjective reports and objective findings raised suspicion of factitious disorder, which coexisted with underlying dementia and psychosis. A multimodal treatment plan including strict glycemic control, cautious use of antipsychotics, and caregiver psychoeducation was implemented.
Conclusion: This case underscores the diagnostic complexity when factitious disorder co-occurs with dementia and psychosis in an elderly patient with uncontrolled diabetes. It highlights the importance of integrating metabolic, neurological, and psychiatric perspectives for accurate diagnosis and effective management. Clinicians should remain vigilant for factitious presentations in older adults, particularly when inconsistencies arise between patient reports and objective findings.

Keywords: Late Onset Dementia, Geriatric, Frontal Lobe, Psychosis

Introduction

Psychiatric symptoms arising de novo in late life warrant careful scrutiny, as they may represent manifestations of underlying neurological pathology.[1] Studies indicate that up to 10–20% of new psychiatric presentations in the elderly can be traced to secondary causes such as neurodegenerative disorders, vascular insults, or space-occupying lesions.[2]
Late-onset psychosis, defined as onset after age 60, is a particularly challenging entity. It is commonly misattributed to depression with psychotic features, late-onset schizophrenia-like psychosis, or dementia-related behavioral disturbances.[3,4] However, neurological conditions such as frontal lobe tumors or cerebrovascular disease may masquerade with predominant affective and psychotic features.[5]
The frontal lobes play a central role in regulating personality, mood, and executive functioning. Lesions in this region are well-documented to cause disinhibition, irritability, affective lability, and delusional ideation, often leading to psychiatric misdiagnosis.[6,7] This is especially problematic in primary care settings, where neuroimaging is not routinely performed and psychiatric referral may be delayed.
In low- and middle-income countries, cultural beliefs and stigma surrounding mental illness further obscure diagnostic pathways.[8] Families may attribute behavioral change in older adults to "normal aging" or stress, contributing to under-recognition of organic causes.[9]
We report a case of a 68-year-old male with psychiatric onset of a frontal lobe lesion, initially misdiagnosed as primary psychiatric illness. The case underscores diagnostic pitfalls and highlights the importance of integrated neuropsychiatric evaluation.

Case Presentation

Patient profile: A 68-year-old retired schoolteacher, right-handed, married, and living with his family in an urban setting, presented with a six-month history of behavioral and cognitive changes. He had no prior psychiatric or neurological illness, no substance use history, and no significant medical comorbidities. Family history was negative for psychiatric disorders.

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Current Medications: Before admission, the patient was on Metformin (500 mg twice daily) and Glimepiride (2 mg once daily) but was poorly compliant.

Chronology of symptoms:

- Month 1–2: The patient exhibited subtle forgetfulness, misplacing household items, and increased irritability. Family noticed he became unusually suspicious, accusing neighbors of theft.
- Month 3–4: Emotional lability emerged, with frequent crying spells alternating with sudden laughter. He withdrew from social activities and showed reduced interest in previously enjoyable tasks. Sleep was fragmented, and appetite declined.
- Month 5–6: Behavioral disturbances worsened; he displayed episodes of agitation, disinhibition (such as making inappropriate jokes), and incoherent speech. Family also observed unsteady gait and mild right arm weakness.

Initial treatment: A general practitioner diagnosed him with late-onset depression with psychotic features and initiated antidepressants (sertraline 50 mg/day) and a low-dose antipsychotic (risperidone 1 mg/day). Minimal improvement was observed after four weeks.

Hospital admission: On admission to our service, the patient was alert but disoriented to time.

Mental Status Examination:

- Appearance: Disheveled, reduced eye contact.
- Speech: Dysarthric, tangential, occasional word-finding pauses.
- Mood/Affect: Labile; alternated between irritability and tearfulness.
- Thought: Persecutory delusions (neighbors stealing items).
- Cognition: Attention span reduced; immediate recall intact, recent memory impaired; poor abstraction.
- Insight/Judgment: Markedly impaired.
- Neurological Examination:
- Cranial nerves: Intact.
- Motor: Right upper limb weakness (4/5).
- Reflexes: Exaggerated deep tendon reflexes on the right.
- Coordination: Mild dysmetria.
- Gait: Broad-based, unsteady.

Investigations

- Routine blood tests: Normal (CBC, LFT, RFT, electrolytes, thyroid profile, B12) (Table 1)
- MRI Brain: T2-weighted images revealed a left frontal lobe lesion (heterogeneous mass with surrounding edema, mass effect on ipsilateral lateral ventricle, possible gliotic changes). No metastatic lesions elsewhere. (Figure 1&2)

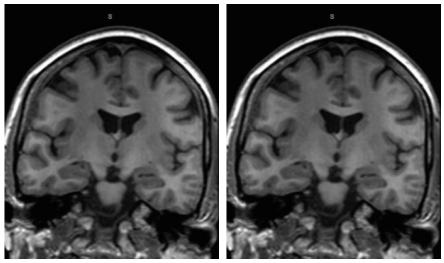
Management:

- Psychiatric care: Risperidone increased to 2 mg/day; short course of lorazepam (1 mg at night) for agitation.
- Medical care: Dexamethasone 4 mg IV q8h initiated to reduce edema.
- Neurosurgical opinion: Surgery was advised, but the family opted for conservative management given age and risks.
- Course and follow-up: Over the next two weeks, irritability and agitation improved with antipsychotic titration and steroids. Cognitive impairment and gait disturbances persisted. The family was counseled extensively about prognosis, supportive care, and potential surgical intervention.

Table 1. Investigations

Parameter	Value	Reference Range	Interpretation
Hemoglobin	13.6 g/dL	13.0–17.0 g/dL	Normal
WBC count	7,800/µL	4,000–11,000/µL	Normal
Platelets	220 $\times 10^9$/L	150–400 $\times 10^9$/L	Normal
Fasting blood glucose	142 mg/dL	70–110 mg/dL	Elevated
HbA1c	8.1%	<6.5%	Poor glycemic control
Thyroid-stimulating hormone	2.1 µIU/mL	0.4–4.5 µIU/mL	Normal
Vitamin B12	390 pg/mL	200–900 pg/mL	Normal

Figure 1: MRI Sagittal and Coronal Views



Discussion

This case illustrates the diagnostic complexity inherent in late-onset psychiatric syndromes and underscores the importance of maintaining a high index of suspicion for secondary causes. Psychiatric illness with onset after the age of 60 is uncommon and frequently suggests an underlying organic etiology rather than a primary psychiatric disorder.[10] Although very-late-onset schizophrenia-like psychosis is a recognized entity, it remains considerably rarer than secondary psychoses arising from neurological or systemic pathology.[11] The frontal lobes play a central role in executive functioning, emotional regulation, and socially appropriate behavior. Lesions affecting this region are well known to present with prominent psychiatric manifestations, often preceding or overshadowing overt neurological deficits. Symptoms such as irritability, affective lability, apathy, disinhibition, and psychosis are commonly described in frontal lobe pathology.[7] The delusional content, behavioral disinhibition, and emotional instability observed in the present patient align closely with this neuropsychiatric profile, contributing to the initial diagnostic challenge.

From a diagnostic standpoint, the clinical presentation demonstrated overlap with several DSM-5-TR categories. Features consistent with Major Neurocognitive Disorder included a significant decline in executive function and behavior that interfered with independent functioning. The persistence of persecutory delusions in the absence of prominent hallucinations raised consideration of Delusional Disorder, persecutory type. Additionally, inconsistencies in symptom reporting and illness behavior briefly suggested Factitious Disorder Imposed on Self. However, the coexistence of progressive neurological signs ultimately favored an organic etiology, highlighting the limitations of applying purely phenomenological psychiatric diagnoses in late-onset presentations.

Several diagnostic red flags were evident in this case, including advanced age at onset, absence of prior psychiatric history, relatively rapid symptom progression over months, and the emergence of neurological signs such as gait disturbance, dysarthria, and motor weakness. These features are well-established indicators of secondary psychosis and should prompt early neuroimaging before attributing symptoms to a primary psychiatric disorder.[12] Failure to recognize these warning signs can result in delayed diagnosis and inappropriate management. The initial misdiagnosis in this case reflects common pitfalls encountered in both primary care and psychiatric settings. In resource-limited environments, clinicians may rely heavily on symptom-based diagnoses due to restricted access to neuroimaging and specialist referral pathways. Even within psychiatric practice, inadequate emphasis on detailed neurological examination can lead to organic causes being overlooked, particularly when psychiatric symptoms dominate the clinical picture.

The case also carries broader global and cultural relevance. In South Asian settings, psychiatric and behavioral changes in older adults are frequently misattributed to “stress,” “aging,” or psychosocial factors, contributing to delayed presentation and intervention.[13] Strengthening collaboration between psychiatry and neurology, improving access to diagnostic imaging, and enhancing public and professional awareness are essential steps toward reducing such delays.

In **conclusion**, this case reinforces that late-onset psychiatric symptoms warrant comprehensive evaluation for underlying neurological causes. Rapid progression, absence of prior psychiatric illness, and subtle neurological abnormalities should be treated as critical red flags. Frontal lobe lesions, in particular, may present predominantly with psychiatric features, misleading clinicians across disciplines. An integrated neuropsychiatric approach, early neuroimaging, and multidisciplinary management are essential to ensure timely diagnosis and optimize patient outcomes.

Patient Consent: Written informed consent was obtained from the patient for the publication of this case report, including all clinical details and images. The patient was assured that his identity would remain confidential.

Conflict of Interest: The authors declare no conflicts of interest related to this case report.

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